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Neuro-Orthopedic Surgery for Equinovarus Foot Deformity in Adults: A Narrative Review

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► **To cite this version:**

Étienne Allart, Nadine Sturbois-Nachef, Marjorie Salga, Charlotte Rosselin, Laure Gatin, et al.. Neuro-Orthopedic Surgery for Equinovarus Foot Deformity in Adults: A Narrative Review. Journal of Foot and Ankle Surgery, 2022, 61 (3), pp.648-656. 10.1053/j.jfas.2021.11.012 . hal-03673614

HAL Id: hal-03673614

<https://hal.science/hal-03673614v1>

Submitted on 16 Jun 2022

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1 **Neuro-orthopedic surgery for equinovarus foot deformity in adults: a narrative**
2 **review**

3
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30
31 **Funding sources:** This work was funded in part by ORPEA-CLINEA and Abbvie-
32 Allergan France.

33
34 **Declaration of interest:** None reported

35
36 **Acknowledgment:** The authors thank David Fraser PhD (Biotech Communication
37 SARL, Ploudalmézeau, France) for editorial assistance.

38
39 **Ethical statement:** NA (this paper being a review, no informed consent or ethical
40 approval were needed)

41
42 **Manuscript characteristics**

43 Word Count: 4923 Abstract: 203

72 **Abstract**

73 Neuro-orthopedic surgery is an alternative to the conservative treatment of spastic
74 equinovarus foot (SEF) in adults. The objective of the present narrative review was to
75 summarize current practice with regard to patient assessment, the choice of treatment,
76 the various neuro-orthopedic procedures, and the latter's outcomes. We searched
77 literature databases (MEDLINE, EMBASE, Cochrane) for original articles or opinion
78 papers on surgical treatment of spastic equinovarus foot in adults. Neuro-orthopedic
79 approaches require a careful analysis of the patient's and/or his/her caregiver needs
80 and thus relevant treatment goals. Surgical planning requires detailed knowledge of
81 impairments involved in the spastic equinovarus foot deformity based on a careful
82 clinical examination and additional information from diagnostic nerve blocks and/or a
83 quantitative gait analysis. Procedures mainly target nerves (neurotomy) and tendons
84 (lengthening, transfer, tenotomy). These procedures reduce impairments (spasticity,
85 range of motion, and foot position), improve gait and walking function, but their impact
86 on participation and personalized treatment goals remains to be demonstrated. Neuro-
87 orthopedic surgery is an effective treatment option for spastic equinovarus foot in
88 adults. However, practice is still very heterogeneous and there is no consensus on the
89 medical strategies to be applied before, during and after surgery (particularly the type
90 of anaesthesia, the need for immobilization, rehabilitation procedures).

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95 **Level of Clinical Evidence : 5**

96

97 **Key Words:** assessment; equinovarus foot; neuro-orthopedics; spasticity; stroke;
98 surgery

99

100

101 **Introduction**

102 Spastic equinovarus foot (SEF) is the most common foot deformity secondary
103 to stroke (1), traumatic brain injury (2) and other central nervous diseases (multiple
104 sclerosis, spinal cord injury, etc.). It interferes with the foot's weight-bearing function,
105 causes instability during the stance phase, and limits foot clearance during the swing
106 phase. The deformity also contributes to kinematic abnormalities in the neighboring
107 joints - particularly knee recurvatum in the stance phase, and stiff-knee gait and limb
108 circumduction in the swing phase (3, 4). Consequently, SEF can severely impair
109 walking, limit activities, increase the need for assistive devices or orthoses, and
110 restrict participation.

111 Several treatments for SEF have been developed. Oral medications (such as
112 baclofen, tizanidine or dantrolene) are non-focal treatments that have demonstrated
113 overall poor efficacy on spasticity and have never been specifically studied in SEF (5,
114 6). In details, oral baclofen slightly improve hypertonia and spasms when used at
115 high doses (>80mg/d) (7, 8), but is frequently associated with side effects (the most
116 frequent being fatigue, in 28% of patients) (9). Focal treatment options include
117 physical therapy, ankle-foot orthoses, and botulinum toxin injections (BTIs). Many
118 types of non-pharmacological physical techniques have been developed (e.g.
119 physical therapy, robotics, physiotherapy, etc), but there is lack of high-quality
120 evidence and none have been specifically applied in SEF (10). The use of ankle foot
121 orthoses (AFOs) can improve the ankle and knee kinematics and kinetics but also
122 energy expenditure in spastic children and adults (11, 12). However, AFOs fail to

123 correct equinus when due to a triceps surae contracture, have a poor efficacy on
124 severe varus deformity (13) and force the patient to wear shoes (thus being a factor
125 of activity limitations, preventing the patient walking barefoot). Although BTIs are
126 particularly effective in relieving muscle spasticity, their impact on active function is
127 less clear - especially when the spasticity is severe (14). Moreover, the dose of toxin
128 is limited, the effects are reversible, and so the injections must be repeated regularly.

129 The goal of neuro-orthopedic surgery is to avoid the limitations of conservative
130 treatments, by proposing a focal, long-lasting treatment, enabling able to act on
131 severe hypertonia and contracture. Neuro-orthopedic procedures target nerves,
132 tendons, joints or bones and in order to improve the balance between agonistic and
133 antagonistic muscles around the ankle joint. Neuro-orthopedic surgery for the
134 treatment of SEF offers great potential but must be considered with regard to the
135 individual patient and the available rehabilitation resources. Surgery also relies on an
136 adequate assessment strategy and the precise definition of treatment targets and
137 goals.

138 In this narrative review, we wanted to summarize the key points of the neuro-
139 orthopedic care of patients with SEF, from pathophysiology to treatments. We
140 particularly discuss (i) the pathophysiology of SEF, (ii) the main factors that prompt
141 the choice of neuro-orthopedic surgery from the patient's perspective and as a
142 function of his/her impairments, (iii) the assessments strategy to define surgical
143 targets and treatment goals, and (iv) the various neuro-orthopedic procedures and
144 their expected outcomes.

145

146 **Patients and Methods**

147 The MEDLINE (via PubMed), EMBASE and Cochrane Central Register of
148 Controlled Trials (CENTRAL) literature databases were searched (from 1980 up until
149 October 15th, 2020) for publications (in English or French) related to any of this review's
150 three objectives, using the following terms in particular: spasticity, equinovarus foot,
151 surgery, neuro-orthopedic surgery, neurotomy, tenotomy, gait, gait analysis,
152 assessment, stroke, brain injury, and spinal cord injury. We included opinion papers or
153 original articles describing factors involved in the decision-making process and the
154 patient's assessment in the specific context of neuro-orthopedic treatment of SEF in
155 adults, and original articles describing surgical techniques and their outcomes; we
156 excluded conference papers and articles that only described techniques without
157 presenting any outcome. The reference lists of the selected articles and other literature
158 known to the review's authors were also searched for relevant publications.

159

160

161 **Results**

162

163

164 Fifty-two articles were retrieved from the literature, among which 19 addressed
165 the decision-making process and the patient's assessment and 33 dealt with surgical
166 procedures

167

168 *1. An Overview of the Pathophysiology of SEF*

169 Upper motor neurons are first-order neurons responsible for initiating and
170 modulating movements. If several descending tracts are involved, the main tract is
171 the pyramidal tract, rising from the motor and premotor cortices to the anterior horn of

172 the spinal cord (15). Lesions of the central nervous system involving this tract at any
173 point of its trajectory give rise to a set of symptoms known as the upper motor neuron
174 syndrome combining paresis, muscle overactivity and abnormal reflexes (16). Central
175 paresis is defined as the inability to voluntarily recruit motor units; it combines motor
176 weakness, impairment of muscle selectivity and muscle fatigue (16). Although the
177 term “spasticity” is often used in the literature to describe all types of muscle
178 overactivity, several subtypes of overactivity have been described; they occur at
179 different moments during the gait cycle. Spasticity is defined as phasic velocity-
180 dependent hypertonia occurring at rest, associated with exaggerated stretch reflex
181 (18). Spastic dystonia is defined as a stretch-sensitive tonic muscle contraction, in
182 the absence of volitional command, and in the absence of phasic stretch of the
183 affected muscle (17); one of its main clinical expressions is the typical attitude of the
184 paretic upper limb at rest in brain injured patients (shoulder adduction, elbow flexion,
185 pronation, wrist and finger flexion). Spastic cocontraction represents inappropriate
186 antagonist recruitment triggered by the volitional command on an agonist in the
187 absence of phasic stretch (17), leading to decrease the power of the agonist muscle.
188 Beyond their differences in terms of clinical expression, these types of hypertonia do
189 not share the same pathophysiological features; if they all result in an involuntary
190 muscle contraction, spasticity is mainly due to spinal mechanisms resulting in an
191 hyperexcitability of the stretch reflex mainly due to an alteration of inhibition, whereas
192 spastic dystonia and cocontraction share a supraspinal origin, i.e. an abnormal
193 supraspinal descending drive (17). This is particularly of interest because treatments
194 acting on the disinhibited reflex at the spinal level (as oral or intrathecal baclofen,
195 neurotomy) will not have a long-lasting effect in spastic dystonia or cocontractions
196 (see also section 4.1). Lastly, antagonist muscle overactivity, agonist paresis and

197 immobilization in short position (e.g. equinus when lying in a bed) give rise to soft
198 tissue rearrangements leading to muscle contracture (16). To sum-up, neuromotor
199 disorders consecutive to UMNS can be modelled as an imbalance between paretic
200 agonistic muscles and overactive or shortened antagonistic muscles which generate
201 resistance to movement (16). In the context of SEF, the equinus deformity results
202 from an imbalance between dorsi- and plantar flexors, whereas the varus deformity
203 results from an imbalance between evertor and invertor muscles) (**Fig. 1**).

204 Along with contracture (in some cases), spasticity of the soleus (SOL),
205 gastrocnemius (GS), tibialis posterior (TP), flexor hallucis longus (FHL) and/or flexor
206 digitorum longus (FDL) is responsible for equinus in stance (13). More rarely, spastic
207 dystonia of these muscles may also contribute to equinus in stance. Equinus in the
208 swing phase may result from (i) paresis of the tibialis anterior (TA) and, to a lesser
209 extent, the extensor hallucis longus (EHL) and extensor digitorum longus, or (ii)
210 contracture and/or spastic co-contraction of the plantar flexors (19).

211 The varus deformity must be also analyzed with regard to the moment when it
212 appears in the gait cycle. During the swing phase, varus is mainly due to activation of
213 the TA (medially inserted in the foot) and weakness of the fibularis muscles. During
214 stance, varus is mainly due to TP or triceps surae overactivity (spasticity or spastic
215 dystonia) or contracture. Cases of varus on initial contact will generally have a cause
216 during the swing phase - even though the stance phase is disturbed. More rarely, the
217 EHL (20) or long toe flexors (21) are responsible for varus in the swing or stance
218 phase, whereas the foot's intrinsic muscles (especially the abductor hallucis and the
219 flexor hallucis brevis (FHB)) may synergize with the TP to generate foot varus in
220 stance and (in many cases) metatarsus adductus (22).

221 Two related conditions must be addressed. Firstly, a reduced plantar flexion
222 moment in the pre-swing phase alters gait speed and knee flexion in the swing phase
223 (23); this impairment may be due to (i) plantar flexor overactivity or contracture until
224 pre-swing or (ii) plantar flexor weakness in the pre-swing phase (23). Secondly, SEF
225 is often associated with claw toe (24) mainly involving the toe flexors (the FDL, flexor
226 digitorum brevis (FDB), quadratus plantae, interossei, FHL, and FHB) and, less
227 frequently, the extensors. These conditions result in pain, discomfort, callosity, and
228 gait limitations. The potential worsening of claw toe after the correction of equinus
229 (via a tenodesis effect) must always be considered when planning surgery (13,
230 25).

231

232 *2. When Should Surgery Be Considered?*

233 Surgery is one of several treatment options for SEF and must therefore be
234 considered as part of an overall treatment plan (13, 26). Its indications and modalities
235 mainly depend on the type of neuromotor disorder involved in the SEF deformity
236 (spinal vs. supraspinal origin as mentioned in section 1, see also section 4 for
237 implications in terms of treatment choice), the severity of associated impairments
238 (proximal neuromotor impairments, sensory impairments, cognitive impairments,
239 etc.), the progressive nature or not of the underlying pathology, or the patient's
240 medical history (particularly the time since onset, the recovery rate, the response to
241 conservative treatments, and the patient's level of motivation) (13, 26). The patient's
242 functional status is also important in the decision process since correction of SEF
243 would not give the same result in an independent high-level walker with minor
244 neuromotor troubles vs. a non-ambulant patient for whom the treatment of SEF
245 would be indicated to facilitate transfers. Treating SEF in patients restricted to

246 wheelchair or beds is also an important question, authors agreeing to treat only if
247 SEF is disabling and not to correct the deformity per se (e.g. for wheelchair
248 installation or shoeing) (13, 25).

249 Almost all the studies (n=30, 91%) reviewed here concerned hemiparetic
250 patients after stroke. The other studies addressed hemiparetic traumatic brain injured
251 patients (27–31), and few bilateral involvement due to spinal cord injury of cerebral
252 palsy (30, 32, 33). Although patients may have some common features, the decision
253 process must take account of specific aspects when considering fixed vs.
254 progressive disorders and single vs. bilateral lower-limb involvement. Only one
255 publication covered the influence of demographic parameters; there was no influence
256 of age and sex on the efficacy of surgery (34). The question of the minimal time
257 interval between onset (brain injury) and surgery is subject to debate: Keenan
258 recommends waiting until the patient has recovered as much as possible (at least 9
259 months) (25), whereas Renzenbrick et al. suggested operating at any time if there is
260 no alternative conservative option and if the SEF deformity greatly reduces the speed
261 of recovery (35). In contrast, the time between onset and surgery did not influence
262 the long-term benefit; hence, surgery might be indicated long after the injury (34).

263 Of the various comorbidities, lower limb arteriopathy is most strongly
264 associated with a risk of poor healing and is sometimes considered to be a
265 contraindication (35). Local skin defects (such as heel pressure-sores) or infections
266 (particularly athlete's foot) could represent temporary contraindications. General
267 cardiovascular co-morbidities, which are particularly frequent in stroke patients, could
268 increase the anaesthetic risk and lead to prefer loco-regional anaesthesia.

269 In terms of indications, it is erroneous to consider that neuro-orthopedic
270 surgery is limited to severe deformities or to treatment goals involving active

271 functions. In fact, surgery is an add-on therapy when conservative treatment is
272 ineffective, insufficiently effective or not lastingly effective (25, 26). More rarely,
273 surgery is a first-line treatment option. It may also help to reduce healthcare costs in
274 the long term by drastically reducing costs due to physical therapy (which can be
275 reduced or stopped) and chemodenervation treatments at a long term (36).

276

277

278

279 *3. Pre-operative Assessment: a Crucial Step in the Surgical Process*

280 Pre-operative assessment is crucial in two respects: (i) evaluation of the
281 patient's treatment goals, and (ii) determination of the treatment plan (i.e. the types of
282 surgical and non-surgical procedures that will be required to meet those goals) (13,
283 26). The assessment also enables the medical team to plan procedures implemented
284 at the time of surgery (the type of anesthesia, pain management, adjunct focal
285 treatments), after surgery (postoperative immobilization and rehabilitation), and also
286 address the prioritization of multiple-step procedures (26). There is a broad
287 consensus on the value of a multidisciplinary assessment that involves not only
288 physiatrists and surgeons but also physiotherapists and anesthesiologists (13, 25,
289 26). Some experts have also suggested first-line remote screening or the
290 combination of a remote consultation with a quantitative gait analysis (35, 37).

291

292 *3.1. Is There a Need to Go Beyond a Clinical Examination?*

293 The clinical examination is the same for surgery vs. other focal treatments;
294 there are no surgery-specific features (Appendix A) (26, 38). The majority of experts
295 have pointed out that there are too few clinical data on the muscle targets to be

296 treated (without overtreatment) and on choice of the type of surgical procedure.
297 Beyond clinical observations per se, there are two main strategies: diagnostic nerve
298 block (DNB) and an instrumented gait analysis (with dynamic electromyography
299 (dEMG) or a quantitative gait assessment). Although these two strategies have
300 different bases, they may be complementary. Their respective frequencies of use
301 mainly depend on local habits (with differences from one region or country to another
302 and from one discipline to another) and ease of access.

303 Diagnostic nerve blocks are intended to temporarily suppress muscle
304 overactivity (especially spasticity) and thus enable the physician to (i) determine
305 which muscles are involved in the SEF deformity (e.g. the respective roles of the
306 SOL and GS in equinus, or the role of the TP in varus), (ii) differentiate between
307 muscle overactivity and contracture, and (iii) predict the results of surgery (especially
308 before neurotomy) (39, 40). Diagnostic nerve block consists in injecting (with
309 electrostimulation or ultrasound guidance) a small dose of local anesthetic near the
310 tibial nerve or selectively near a motor branch (41, 42). Given that muscle spindle
311 afferents are preferentially sensitive to local anesthetics, the effects of DNB are
312 typically clear for spasticity and clonus but often less so for other forms of muscle
313 overactivity (43). The DNB procedure is inexpensive and safe (40) but is limited to
314 nerves that can be accessed easily. Intramuscular blocks may be an alternative
315 when a selective nerve block is technically difficult or impossible to achieve.

316 Gait analysis and dEMG also complement a comprehensive clinical
317 examination. Fuller et al. showed that an instrumented gait analysis resulted in a
318 refinement of surgical plan in 64% of patients, independently of the evaluator's
319 experience (44). Furthermore, dEMG used alone is of particular value for studying
320 the factors causing varus deformity (45, 46), and for demonstrating that the activity

321 patterns of muscles having undergone tendon surgery remained unchanged – this is
322 of interest when a palliative transfer is considered (45). Even though these
323 techniques provide large amounts of information, they are not widely available.

324

325 *3.2. Goal Setting*

326 It is now widely acknowledged that the treatment of spasticity must be based
327 on well-defined goals rather than measurements of muscle overactivity *per se* (47);
328 this is particularly true for surgery because the patient's and/or family's expectations
329 may be high (26, 48). Goals must be complaint-driven and SMART, i.e. specific,
330 measurable, attainable (unattainable goals must be discarded), relevant (in the
331 patient's daily life of the) and time-bound. It is important to spend time explaining to
332 the patient what is expected, possible and unattainable and to mutually agree on
333 goals. Specific assessment tools (such as the Goal Attainment Scale, GAS) can help
334 to define goals and quantify changes after the treatment of SEF. However, this goal-
335 centred approach has not been extensively studied, since only Deltombe et al.
336 reported results in SEF, showing its particular interest to assess activities and
337 participation, two domains where usual assessments tools (such as gait endurance,
338 functional independence scores) failed to demonstrate any change contrary to the
339 GAS (48).

340

341 *4. Surgical Procedures*

342

343 *4.1. Procedures for Releasing Overactive/Shortened Antagonistic Muscles*

344 These techniques are based on tendon or nerve procedures. Tendon
345 procedures may be indicated in cases of muscle contracture but also for reducing

346 spasticity in non-shortened muscles because lengthening a tendon shifts the
347 threshold of the neuromuscular spindle (49). However, tendon procedures do not
348 suppress the abnormal descending influx (except in cases of complete tenotomy),
349 which might explain the persistence of muscle overactivity and deformity in some
350 instances (particularly in cases of spastic dystonia). They might also weaken the
351 lengthened muscle (50). The technique of choice depends on the expected
352 lengthening, the status of the antagonists, and the treatment goals: intramuscular
353 lengthening, lengthening of the tendon *per se* ("Z", percutaneously, or with sutures)
354 or simple tenotomy (percutaneous or open) (26).

355 Neurotomy (also called selective partial neurectomy) consists in a 50 to 90%
356 resection of selected motor branches. It produces a strong, long-lasting decrease in
357 muscle spasticity without inducing long-term motor weakness of the target muscles
358 (31, 51). It is important to note that if the neurotomy involves a section of efferent
359 (alpha motoneuron) fibers and afferent (Ia) fibers, the two types of fibers sprout in
360 opposite directions; if motoneurons sprout, afferent fibers not; this explains the
361 reduction in spasticity associated with a long-lasting decrease in the maximum
362 amplitude of the H reflex to maximum amplitude of the M-response (H_{max}/M_{max} ratio)
363 (43), which represent an electrophysiologic marker of the intensity of the spastic
364 reflex. These observations also suggest that neurotomy is effective for spasticity
365 (which mainly involves the stretch reflex at the spinal level) but not for non-reflex
366 overactivities (such as spastic dystonia or co-contractions) that mainly involve
367 abnormal supra-spinal drives (19, 52). This dichotomy is not relevant for tendon
368 procedures that treat hypertonia at the muscle level (as botulinum toxin also does).

369

370 To treat SEF, tendon lengthening and nerve procedures can be applied to any
371 muscle in the leg's posterior compartment (**Fig. 1A**). With regard to the triceps surae,
372 it is essential to differentiate between contracture of the gastrocnemius vs. the soleus
373 because either the gastrocnemius aponeurosis alone or the whole Achilles tendon
374 complex can be lengthened (13). If the value of nerve procedures is not debated in
375 spastic only muscle, the value of nerve and tendon procedures for overactive,
376 shortened muscle is still subject to debate. Indeed, some experts state that tendon
377 lengthening is sufficient because it treats both muscle spasticity and contracture (13,
378 25, 26), whereas others stipulate that long-term effectiveness requires the treatment
379 of spasticity (with neurotomy) and tendon lengthening (51). The use of percutaneous
380 tenotomy (using a needle or a scalpel) in an indication of SEF has not been well
381 documented (53, 54). Lastly, treatment or prevention of claw toe (which can appear
382 after equinus reduction) is based on tendon procedures (intramuscular lengthening or
383 tenotomy of the FDL, FHL or intrinsic muscles) rather than neurotomy (13, 51).

384

385 *4.2. Procedures for Rebalancing Agonists vs. Antagonists: a Focus on the Varus* 386 *Deformity*

387 Most of the cases of varus deformity during the swing phase of gait are due to
388 an imbalance between invertor muscles (the TA, EHL, or FHL) and evertors (the
389 peroneus muscles). Transferring all or part of a distal tendon to a more laterally
390 located insertion point may rebalance the foot's position in the frontal plane (**Fig. 1B**).
391 Split anterior tibial tendon transfer (SPLATT) to the peroneus brevis or the cuboid
392 has been most widely studied (34, 55–58), although some surgeons have also
393 suggested transferring the EHL to the 4th metatarsal bone (59) or fixing the peroneus
394 brevis distal tendon to the tibialis anterior (the Bardot procedure) (60). When the

395 varus deformity occurs in stance phase, the TP is the main target for neurotomy
396 (when fully corrected after DNB) (13, 39), tendon lengthening or tenotomy. Medial
397 intrinsic muscles (the abductor hallucis and FHB) can be targeted in rare cases but
398 the efficacy of this approach has not been reported.

399

400 *4.3. Procedures Aiming at Strengthening Agonistic Muscles in the Swing or Stance* 401 *Phase*

402 Tendon transfers may help to strengthen agonistic muscles, i.e. the plantar
403 flexors in pre-swing phase and the dorsiflexors during the swing phase. The graft can
404 be a co-agonist (which may have the same activation pattern as the weakened
405 muscle) or an antagonist that is rerouted to act as an agonist. In the latter case, the
406 transferred muscle may be less effective in its new role than when the transfer is
407 performed in an indication of peripheral foot drop, and the transfer may mainly act
408 through tenodesis effect (61).

409 To improve dorsiflexion during the swing phase, antagonists (the TP (61, 62)
410 or long toe flexors (63)) can be transferred to the dorsum of the foot (**Fig. 1A**). To
411 improve plantar flexion during the pre-swing phase, it has been suggested that the
412 FHL or the FDL can be transferred to the os calcis (34, 55, 56).

413

414 *The Utility of Joint and Bone Procedures*

415 In adults, joint and bone procedures for SEF are of limited utility. In contrast to
416 disorders acquired in childhood, there are no growth issues. Furthermore, deformities
417 - even very severe ones - can mostly be corrected with tendon surgery. In rare cases
418 of capsular retraction or if tendon procedures fail to fully reduce the varus or valgus
419 deformity, hindfoot and/or midfoot arthrodesis can be considered (64).

420

421 *5. What Can we Expect From Neuro-orthopedic Surgery?*

422

423 Our search of electronic literature databases identified 113 publications, 33 of
424 which presented efficacy results (covering a total of 1010 participants) and were
425 included in the present review. Seventeen publications referred to nerve procedures
426 (n=371 participants) (**Table 1**) (27–31, 33, 39, 43, 51, 65–72), 12 referred to tendon
427 procedures (n=527) (**Table 2**) (21, 34, 53, 55–57, 59, 61, 62, 62, 63, 73) and four
428 referred to mixed tendon and nerve procedures (n=112) (**Table 3**) (48, 74–76). There
429 were only four comparative studies, one randomized controlled trial (comparing tibial
430 nerve neurotomy with BTI) (65), and three parallel group studies on tendon
431 procedures (21, 56, 59). Impairments were frequently assessed and were considered
432 as the primary outcome in all the reviewed studies. Activity limitations were evaluated
433 in 26 publications, restriction of participation was evaluated in 2 and patient
434 satisfaction was evaluated in 12.

435

436 *5.1. Nerve Procedures*

437 The nerve procedures were intended to treat equinus (targeting branches of
438 the SOL or the GS) and or varus mainly in stance (branches of the TP and correction
439 of the equinus), whereas some surgeons also sought to treat claw toe by targeting
440 fibers of the FHL or FDL. The sample sizes of studies dealing with neurotomy were
441 relatively small (mean: 21.8 participants) but the studies were homogeneous in that
442 they all covered tibial nerve neurotomies (TNNs).

443 The publications reported that TNN reduced the patients' impairments (**Table**
444 **1**). Muscle tone was reduced in a lasting manner, and the passive range of motion in

445 dorsiflexion was increased in 15 studies. Nerve procedures improved dorsal flexor
446 strength in 12 studies but also induced transient weakness of the plantar flexors in 6
447 studies; the weakness resolved in long-term assessments (1 to 2 years after
448 surgery). Foot position (especially during the stance phase) was found to be
449 improved in 12 studies, while knee recurvatum was found to be improved in 7. Lastly,
450 TNN was associated with variable, small improvements in spatiotemporal gait
451 parameters.

452 With regard to activities, the use of an ankle foot orthosis was reduced or
453 discontinued after surgery in 8 studies. Balance and walking (walking speed,
454 independent walking scores, and walking aid use) also improved in 8 studies. Only
455 Bollens et al. assessed the impact of TNN on participation and quality of life but
456 found no effect (65). Surgery was associated with greater patient satisfaction in 7
457 studies.

458

459 *5.2. Tendon Procedures*

460 The sample sizes in studies of tendon procedures were larger (mean: 43.9
461 participants). However, the surgical techniques varied markedly and were often
462 combined with other procedures during the same surgical session – making it difficult
463 to assess the effect of a specific procedure. All the studies sought to correct
464 equinovarus, 4 sought to improve dorsiflexion in the swing phase (i.e. to correct drop
465 foot) (21, 61–63) and 3 sought to reinforce plantar flexion in the pre-swing phase (34,
466 55, 56) (**Table 2**). The assessment of impairments was mostly limited to the passive
467 and/or active ranges of motion and the qualitative foot position. Gait parameters were
468 more rarely assessed. Although walking activities and the need for assistive devices
469 were frequently evaluated, participation was never assessed. Overall, tendon

470 procedures were associated with drastic improvements in foot position; however, it
471 was not possible to differentiate between effects on equinus vs. varus deformities.
472 Gait parameters were improved in about two thirds of the studies. The requirement
473 for an ankle-foot orthosis was less frequent after surgery in all but one study, and
474 walking ability improved in all studies. The levels of patient satisfaction were high in
475 the five studies that evaluated this variable.

476 The varus deformity was most frequently treated with the SPLATT procedure
477 (34, 55–58), with good functional outcomes. The combination of an anterior transfer
478 of the FHL to the dorsum midfoot with SPLATT did not give any additional benefit
479 (57). Lateral transfer of the entire TA tendon gave worst results (in terms of foot
480 position and drop foot in the swing phase) than FDL and FHL transfer to the dorsal
481 midfoot; the latter operation corrected the varus deformity and improve dorsiflexion in
482 the swing phase (see below) (21). In patients who had a varus deformity in the swing
483 phase but did not exhibit overactivity of the TA, an EHL transfer to the 4th metatarsal
484 bone gave the same results as SPLATT in patients with an overactive TA (59).
485 Lastly, it must be borne in mind that the correction of equinus in stance helps to
486 correct the varus deformity. However, the respective impacts of these procedures
487 cannot be assessed when they are performed together.

488 It is difficult to assess the efficacy of specific procedures aimed at improving
489 foot drop in the swing phase (i.e. muscle transfers to the dorsum of the foot).
490 Although positive trends are noted in the various studies (21, 61–63), each also
491 included calf muscle procedures for the correction of equinus in stance (Achilles
492 tendon or gastrocnemius aponeurosis lengthening) - a factor that is per se likely to
493 improve dorsiflexion in the swing phase (even without muscle transfer). Furthermore,
494 the transfer may act more through a “passive” tenodesis effect than through “active”

495 reintegration of the transfer (61). Unexpectedly, these procedures (especially for TP
496 transfer) tended to result in pes cavus rather than a flat foot (61–63).

497 Lastly, in a study of the additive effect of FDL and FHL transfer to the os calci
498 for equinovarus correction (in order to improve plantar flexion in the pre-swing
499 phase), Keenan et al. found a trend towards greater gait speed, better walking
500 function, and decreased use of orthoses (56). The two other studies did not feature a
501 control group and so no conclusions can be drawn with regard to the value of toe
502 flexor transfer.

503

504 5.3. *Mixed Procedures*

505 Mixing nerve and tendon procedures in the same surgical time would allow to
506 garner the best advantage of each type of techniques. Nevertheless, very few studies
507 reported such an approach (**Table 3**) . This can be explained by the lower diffusion
508 of the practice of nerve gestures, by different habits between disciplines (nerve
509 gestures being more often performed by neurosurgical teams, tendon gestures by
510 orthopedic teams), and finally by difficulties to identify causal relationship from
511 various gestures. Some authors also advised to perform the tendinous procedure
512 after the nervous one because the latter could be followed by an increase in muscle
513 contracture (51, 72), but this seems to be questioned (56).

514

515 6. *Limits*

516 The present narrative review aimed at offering an overview the main features
517 of the neuro-orthopedic approach of equinovarus foot from pathophysiology to
518 treatments. A systematic review would have better emphasized treatment outcome,
519 but it would have suppressed the didactic dimension of the paper. Such a systematic

520 review have been already performed for nerve procedure (77); the wide
521 heterogeneity in surgical procedures and outcomes make it difficult to perform for
522 tendon procedures. Lastly, only case series and single-center studies have been
523 reported to date, limiting possibilities to draw strong conclusions.

524

525 **Discussion**

526 Neuro-orthopedic surgery is a powerful treatment option for SEF in adults. It
527 requires a careful assessment of the patient's treatment goals. The surgical plan
528 should be built on both clinical data and additional assessments (DNB or gait
529 analysis). Several different types of surgical techniques have been described. Most
530 procedures target nerves (neurotomies) and tendons, aiming to correct equinus in
531 the stance and/or swing phases or correcting varus. Although these procedures
532 resulted in a reduction in impairment (particularly for spasticity, range of motion, foot
533 position and, to a lesser extent, gait) and greater walking capacities, future studies
534 must seek to demonstrate an impact on participation and the achievement of
535 personal treatment goals.

536 Our review also highlighted gaps in the research data and topics that are
537 subject to debate. First, surgical practice is very heterogeneous. Secondly, only case
538 series and single-center studies have been reported to date. Thirdly, the use of DNB
539 vs. instrumented gait analysis as an additional assessment and the relative value of
540 nerve vs. tendon procedures must be addressed. Fourthly, there is no consensus on
541 the relationship between surgical and other treatment strategies on one hand and
542 medical strategies before and after surgery (the type of anesthesia, immobilization (if
543 needed), and rehabilitation) on the other. In the future, there is a need for a broad,

544 international, multidisciplinary expert consensus on the assessment and neuro-
545 orthopedic treatment of SEF.

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778 **Figure and Tables legends**

779 **Figure1.** The causes of and main neuro-orthopedic treatment options for equinus (A)
780 and varus (B) deformities.

781 *l: tendon lengthening; t: tenotomy; AT: Achilles tendon; EHL: extensor hallucis*
782 *longus; FDL: flexor digitorum longus; FHL: flexor hallucis longus; GS: gastrocnemius;*
783 *SOL: soleus; SPLATT: split anterior tibial tendon transfer; TNN: tibial nerve*
784 *neurotomy; TP: tibialis posterior*
785

786 **Table 1.** Outcomes of nerve procedures

787 *∅: no statistically significant difference; ↑: increase; ↓: decrease; *: mean follow-up; £:*
788 *max follow-up*
789 *AS: Ashworth Scale; BTI: botulinum toxin injections; FAC: Functional Ambulation*
790 *Classification; FDL: flexor digitorum longus; FHL: flexor hallucis longus; GS:*
791 *gastrocnemius; MAS: Modified Ashworth Scale; NT: not tested; PROM: passive*
792 *range of motion; RMA: Rivermead Motor Assessment; SF36: Short Form Health*
793 *Survey (36 items); SOL: soleus; SR: stretch reflex; TNN: tibial nerve neurotomy; TP:*
794 *tibialis posterior*
795

796 **Table 2.** Outcomes of tendon procedures

797 *The colours on the left indicate the studies' treatment objectives. Green = correction*
798 *of equinovarus foot; brown = correction of foot drop; blue = improvement of plantar*
799 *flexion in the pre-swing phase.*
800 *l: tendon lengthening; t: tenotomy, tr: tendon transfer, pc: percutaneous;*
801 *∅: no statistically significant difference; ↑: increase; ↓: decrease; *: mean follow-up; £:*
802 *max follow-up*
803 *AS: Ashworth Scale; AT: Achilles tendon; EHL: extensor hallucis longus; FDL: flexor*
804 *digitorum longus; FHL: flexor hallucis longus; GS: gastrocnemius; MAS: Modified*
805 *Ashworth Scale; NT: not tested; ROM: passive range of motion; SOL: soleus;*
806 *SPLATT: split anterior tibial tendon transfer; SR: stretch reflex; TP: tibialis posterior*
807

808 **Table 3.** Outcomes of mixed procedures

809 *l: tendon lengthening; t: tenotomy; tr: tendon transfer*
810 *∅: no statistically significant difference; ↑: increase; ↓: decrease; *: mean follow-up; £:*
811 *max follow-up*
812 *AT: Achilles tendon; FDL: flexor digitorum longus; FHL: flexor hallucis longus; GS:*
813 *gastrocnemius; MAS: Modified Ashworth Scale; NT: not tested; PROM: passive*
814 *range of motion; SOL: soleus; SPLATT: split anterior tibial tendon transfer; SR:*
815 *stretch reflex; TP: tibialis posterior*
816

817

818

A

Plantarflexors

Stance

SOL, GS ± TP, FDL, FHL

Spasticity *TNN*
AT-I or GS aponeurotomy
TP, FDL or FHL I or t

spastic dystonia ± *TNN ?*
AT-I or GS aponeurotomy
TP, FDL or FHL I or t

Contracture *AT-I or GS aponeurotomy*
TP, FDL or FHL I or t
Associate TNN ?

Swing

SOL, GS ± TP, FDL, FHL

Spastic cocontractions -

Contracture *AT-I or GS aponeurotomy*
TP, FDL or FHL I or t
Associate TNN ?



Dorsiflexors

Swing

TA ± EHL, EDL Paresis

Anterior transfer of the TP or the FDL/FHL to the dorsal midfoot

B

Invertors

Stance

TP spasticity *TP-TNN, TP-I or t*

EHL dystonia *EHL-t (4th metatarsal bone)*

Intrinsic hallux muscles (AH and FHB) -

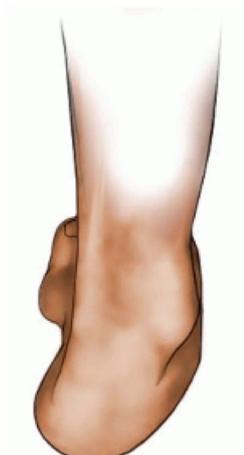
Equinus in stance *see (A)*

Varus at initial contact (in swing)

Swing

TA and EHL unbalanced activity

SPLATT
EHL-t



Evertors

2 phases: Peroneus muscles paresis

Table 1. Outcomes of nerve procedures

| Authors | Targeted muscles | Follow-up | Body structure and function | | | | | | | Activities | | Participation (SATISPART, SF-36) | Patient satisfaction | Goal attainment | |
|-----------------------------|-------------------------|-------------------|-----------------------------|------|-----------------|-----------------|---------------|-----------------|--------------------------------|--------------|------------------------------|-------------------------------------|----------------------|-----------------|----|
| | | | Muscle tone | PROM | Muscle strength | | Foot position | Knee recurvatum | Spatiotemporal gait parameters | Orthosis use | Walking abilities | | | | |
| | | | | | Dorsal flexors | Plantar flexors | | | | | | | | | |
| Bollens et al., 2013 (65) | SOL ± TP and FHL vs BTI | 6 M [£] | ↓ MAS | ∅ | ∅ | ∅ | ∅ | ∅ | ∅ | NT | NT | ∅ ABILOCO | ∅ (SATISPART, SF-36) | NT | NT |
| Buffenoir et al., 2004 (29) | SOL-GS ± TP-FHL-FDL | 10 M* | ↓ SR | ↑ | NT | NT | ↑ | ↓ | NT | ↓ | ↑ endurance | NT | ↑ | NT | |
| Buffenoir et al., 2013 (30) | SOL ± GS-TP-FDL | 15 M [£] | ↓ MAS, SR | ↑ | NT | NT | ↑ | ∅ | NT | NT | ↑ independent walking scores | NT | NT | NT | |
| Caillet et al., 2003 (66) | SOL-GS-TP-FHL | 6 M [£] | ↓ MAS | ↑ | ↑ stance | ∅ | ↑ | ↓ stance | ∅ | NT | NT | NT | ↑ (gait discomfort) | NT | |
| Decq et al., 2000 (33) | SOL ± GS-TP-FDL-FHL | 15 M* | ↓ MAS | ↑ | NT | NT | ↑ | ↓ | ∅ | ↓ | NT | NT | ↑ | NT | |
| Deltombe et al., 2006 (67) | SOL-GS-FHL | 2 Y [£] | ↓ MAS | ↑ | ↑ | NT | ∅ | ↓ | NT | ↓ | NT | NT | NT | NT | |
| Deltombe et al., 2008 (43) | SOL ± GS-TP-FHL | 1 Y [£] | ↓ MAS | ↑ | NT | ↓ | ↑ stance | ∅ | ∅ | NT | NT | NT | NT | NT | |
| Deltombe et al., 2010 (31) | SOL ± GS-TP-FHL | 2 Y [£] | ↓ AS | ↑ | ↑ | ↓ | ↑ | ∅ | ↑ speed | NT | NT | NT | NT | NT | |
| Deltombe et al., 2015 (39) | ± SOL-GS-TP-FHL | 2 Y [£] | ↓ MAS | ↑ M2 | ↑ | ↓ | ↑ | ∅ | ↑ speed | NT | NT | NT | NT | NT | |
| Fève et al., 1997 (27) | SOL-GS | 1 M [£] | ↓ SR | ∅ | ↑ | ↓ | NT | NT | ∅ | ↓ | ∅ | NT | NT | NT | |

| | | | | | | | | | | | | | | |
|---------------------------------------|----------------------------|-------------------|---------------|------|------|------|----------|----|-----------------|-----------|--------------------|----|-----------|----|
| Fouad et al., 2011 (68) | SOL-GS-TP ± FDH-FHL | 2Y* | ↓AS | NT | ↑ | ↑ | NT | NT | NT | NT | NT | NT | NT | NT |
| Jang et al., 2004 (69) | SOL-GS-TP | 12 M [£] | ↓ | ↑ | ↑ | NT | NT | NT | NT | ↓ | NT | NT | NT | NT |
| Kim et al., 2010 (70) | SOL-GS | 36.7 M* | ↓AS | ↑ | NT | NT | NT | NT | NT | NT | NT | NT | ↑ | NT |
| Le Bocq et al., 2016 (71) | SOL-GS ± TP | 5 M [£] | ↓MAS | ↑ | ↑ | NT | ↑ | ∅ | ↑ | ↓ | ↑ walking function | NT | ↑ | NT |
| Roujeau et al., 2003 (60) | SOL-GS ± TP-FHL-FDL | 28 M* | ↓SR | ↑ | ↑ | NT | ↑ | ↓ | NT | NT | NT | NT | NT | NT |
| Rousseaux et al., 2008 (72) | SOL-GS ± TP-FHL-FDL vs BTI | 1 Y [£] | ↓MAS, TNN>BTI | ↑TNN | ↑TNN | ↓TNN | ↑TNN | ↓ | ↑ speed TNN>BTI | ↓ TNN>BTI | ↑ FAC, RMA | NT | ↑ TNN>BTI | NT |
| Rousseaux et al., 2009 (51) | SOL-GS ± TP-FDL | 2 Y [£] | ↓MAS | ↑ | | ↓ | ↑ stance | ↓ | ↑speed | ↓ | ↑ FAC, RMA | NT | ↑ | NT |

∅: no statistically significant difference; ↑: increase; ↓: decrease; *: mean follow-up; £: max follow-up

AS: Ashworth Scale; BTI: botulinum toxin injections; FAC: Functional Ambulation Classification; FDL: flexor digitorum longus; FHL: flexor hallucis longus; GS: gastrocnemius; MAS: Modified Ashworth Scale; NT: not tested; PROM: passive range of motion; RMA: Rivermead Motor Assessment; SF-36: Short Form Health Survey (36 items); SOL: soleus; SR: stretch reflex; TNN: tibial nerve neurotomy; TP: tibialis posterior

Table 2. Outcomes of tendon procedures

| Authors | Intervention | Follow-up | Body structures and functions | | | Activities | | Participation | Satisfaction |
|-----------------------------------|-----------------------------------------------------------------------------------------------------|-------------------|-------------------------------|---------------|----------------|--------------|-------------------|---------------|--------------|
| | | | PROM | Foot position | Gait | Orthosis use | Walking abilities | | |
| Boffeli et al., 2014 (53) | pc-AT-l + TP-l + pc-FDL-t | 22 M [£] | a-p-↑ | ↑ | NT | ↓ | ↑ | NT | NT |
| Carda et al., 2010 (59) | EHL-tr or SPLATT | 1 Y [£] | a-↑ | ↑= | ↑= | ↓= | ↑= | NT | NT |
| Giannotti et al., 2019 (73) | AT-l, FDL-t (± SPLATT, FHL and EHL-tr) | 1Y [£] | a-↑ | ↑ | ↑ | NT | NT | NT | |
| Keenan et al., 1984 (57) | SPLATT (n=54) | 49.7 M* | NT | ↑= | NT | ↓= | ↑= | NT | NT |
| Vogt et al., 2011 (58) | SPLATT ±AT-l and pc-FDL-t | 65 M* | NT | NT | ∅ | ↓ | ↑ | NT | ↑ |
| Edwards et al., 1993 (55) | SPLATT + AT-l (± TP-l, FDL-t, FHL-tr, triple arthrodesis) | 4Y [£] | NT | ↑ | ↑ | ↓ | ↑ | NT | NT |
| Keenan et al., 1999 (56) | (I) control group: SPLATT + pc-AT-l + FDL-t (II) study group: same + FDL and FHL-tr to os calcis | 41M* 24M* | NT | ↑ | ↑velocity (II) | ↓ (II) | ↑ | NT | NT |
| Namdari et al., 2009 (34) | SPLATT ± FDL-tr to os calcis, AT-l | 50.1 M* | NT | ↑ | NT | ↓ | ↑ | NT | ↑ |
| Gasq et al., 2013 (62) | TP-t to dorsal midfoot + AT-l ± FDL-t | 57.9 M* | NT | ↑ swing | ∅ | ↓ | ∅ | NT | ↑ |
| Morita et al., 1998 (21) | AT-l + Anterior FDL/FHL-tr (I) or TA-tr (II) | 33 M* | NT | ↑ (> I) | NT | ↓ (> I) | ↑= | NT | NT |
| Ono et al., 1980 (63) | FDL and FHL-tr to dorsal midfoot + AT-l | 4 to 8 Y | NT | ↑ | NT | ∅ | NT | NT | ↑ |
| Sturbois-Nachef et al., 2019 (61) | TP-t to dorsal midfoot ± AT-l, FDL-r, FHL-l, SOL-GS neurotomy | 5.5 Y* | ↑ | ↑ | NT | ↓ | ↑ | NT | ↑ |

The colours on the left indicate the studies' treatment objectives. Green = correction of equinovarus foot; brown = correction of foot drop; blue = improvement of plantar flexion in the pre-swing phase.

l: tendon lengthening; t: tenotomy, tr: tendon transfer, pc: percutaneous;

∅: no statistically significant difference; ↑: increase; ↓: decrease; *: mean follow-up, £: max follow-up

AS: Ashworth Scale; AT: Achilles tendon; EHL: extensor hallucis longus; FDL: flexor digitorum longus; FHL: flexor hallucis longus; GS: gastrocnemius; MAS: Modified Ashworth Scale; NT: not tested; ROM: passive range of motion; SOL: soleus; SPLATT: split anterior tibial tendon transfer; SR: stretch reflex; TP: tibialis posterior

Table 3. Outcomes of mixed procedures

| Authors | Intervention | Follow-up | Body structures and functions | | | | | Activities | | Participation |
|-----------------------------------------|--------------------------------------------------------------------------------|------------------|-------------------------------|------|--------------------|-----------------|-----------------|--------------|--------------------|---------------|
| | | | Spasticity | PROM | Foot position | Muscle strength | knee recurvatum | orthotic use | walking capacities | |
| Bleyenheuft et al., 2008 (74) | SOL neurotomy ± GS-l | 7 to 19 M | ↓ | ↑ | NT | NT | NT | NT | NT | NT |
| Deltombe et al., 2019 (48) | Depending on the patient: SOL-GS-TP-FHL neurotomy, AT-l, FHL or FDL-tr, SPLATT | 1 Y [£] | ↓ | ↑ | ↑swing and stance | NT | ∅ | ↓ | ∅ | ∅ |
| Khalil et al., 2016 (75) | SOL-GS-TP neurotomy ± AT-l, FDL-t | 10.7 M* | NT | NT | ↑ baropodometry | NT | NT | NT | NT | NT |
| Sitthinamsuwan et al., 2012 (76) | SOL-GS ± TP neurotomy ± AT-l | 6 M [£] | ↓ | ↑ | ↑ | ↑ | NT | NT | ↑ | NT |

l: tendon lengthening; t: tenotomy; tr: tendon transfer

*∅: no statistically significant difference; ↑: increase; ↓: decrease; *: mean follow-up; £: max follow-up*

AT: Achilles tendon; FDL: flexor digitorum longus; FHL: flexor hallucis longus; GS: gastrocnemius; MAS: Modified Ashworth Scale; NT: not tested; PROM: passive range of motion; SOL: soleus; SPLATT: split anterior tibial tendon transfer; SR: stretch reflex; TP: tibialis posterior